

The opinion in support of the decision being entered today was not written for publication and is not binding precedent of the Board.

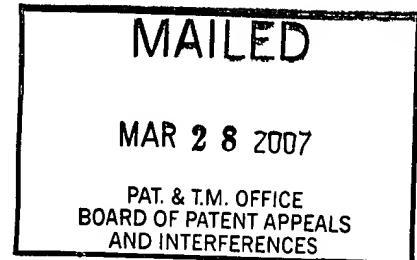
## UNITED STATES PATENT AND TRADEMARK OFFICE

### BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Ex parte GREGORY DONOHO, JOHN SCOVILLE,  
C. ALEXANDER TURNER, JR., GLENN FRIEDRICH,  
BRIAN ZAMBROWICZ, and ARTHUR T. SANDS

Appeal 2004-1103  
Application 09/733,387

ON BRIEF



Before SCHEINER, ADAMS, and GRIMES, Administrative Patent Judges.

ADAMS, Administrative Patent Judge.

#### DECISION ON REQUEST FOR REHEARING

Appellants request reconsideration (rehearing) of the Board's June 30, 2004 Decision, affirming the rejection of claim 1 under 35 U.S.C. § 101 as lacking utility and § 112, first paragraph, for lack of enablement based on the finding of lack of utility. Claims 2, 3, and 6-9 fell together with claim 1.<sup>1</sup>

Claim 1 is illustrative of the subject matter on appeal and is reproduced below:

1. An isolated nucleic acid molecule comprising at least 22 contiguous bases of nucleotide sequence from SEQ ID NO:43.

<sup>1</sup> Having disposed of all claims on appeal, we did not reach the merits of the separate rejection of claim 1 under both the enablement and written description provisions of 35 U.S.C. § 112, first paragraph.

According to appellants (Request, page 2, emphasis removed),

[g]iven the scientific evidence of record, there can be no question that those skilled in the art would clearly believe that [a]ppellants' sequence is a functional protein, as opposed to the assertion by the [e]xaminer that "there is no sufficient and credible information that indicates the published sequence is truly function GPCR" . . . .

In support of this assertion, appellants' Request raises five points which appellants believe the Board failed to consider in reaching its Decision. Id. We will take each in turn.

1. Appellants assert (id., emphasis removed) that "[s]equences sharing between 90-100% percent identity at the protein level over the entire length of the claimed sequence are present in the leading scientific repository for biological sequence data (GenBank)." We note, however, that simply demonstrating that sequences sharing some degree of identify with appellants' disclosed sequence are known in GenBank does nothing to establish a utility for the invention of claim 1. Accordingly, without more, we are not persuaded by this argument.

2. Apparently, recognizing that the GenBank sequence data alone is insufficient to establish a utility for the invention of claim 1, appellants direct attention to five post-filing date references (see Brief, Exhibits A-E). Request, page 2. According to appellants (id.), these references demonstrate that "third party scientists" annotated the GenBank sequences as G protein-coupled receptors.

While we would agree that post-filing date references can be used as evidence of the level of ordinary skill in the art at the time of the application,<sup>2</sup> appellants have not demonstrated that at the time of appellants' claimed invention a person of ordinary skill in the art would have recognized that these GenBank sequences demonstrate that appellants' claimed sequence is a functional G protein-coupled receptor. At best, appellants have demonstrated

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<sup>2</sup> In re Hogan, 559 F.2d 595, 605, 194 USPQ 527, 537 (CCPA 1977) ("This court has approved use of later publications as evidence of the state of the art existing on the filing date of an application." (footnote omitted)).

that after their filing date, sequences with some degree of similarity to their own have been annotated as G protein-coupled receptors. Appellants have not, however, demonstrated that their protein is a G protein-coupled receptor. For the reasons set forth in our June 30, 2004 Decision, appellants have not established a utility for the sequence of claim 1. Later filed publications cannot be used to supplement an insufficient disclosure in an earlier application to render it enabling. In re Glass, 492 F.2d 1228, 1232, 181 USPQ 31, 34 (CCPA 1974) (later publications which add to the knowledge of the art cannot be used to supplement an insufficient disclosure). “It is an applicant’s obligation to supply enabling disclosure without reliance on what others may publish after he has filed an application on what is supposed to be a completed invention. If he cannot supply enabling information, he is not yet in a position to file.” Glass, 492 F.2d at 1232, 181 USPQ at 34. While the Glass court addressed the enablement requirement of § 112, the same rule applies to the utility requirement of § 101. In re Brana, 51 F.3d 1560, 1567 n.19, 34 USPQ2d 1436, 1441 n.19 (Fed. Cir. 1995).

3. Apparently, recognizing the deficiency in their argument regarding the post-filing date references, appellants direct our attention to a murine sequence that shares 68% identity and 78% similarity at the amino acid level of the entire length of the sequence described in appellants’ specification and is present in GenBank. Request, page 2. Again, sequence listings alone cannot establish a utility for the invention of claim 1.

4. According to appellants (id.), “third party scientists” annotated the murine sequence present in GenBank as “*Mus musculus Pb99 [(Pb99)] gene sequence*”. See Brief, Exhibit F. This annotation, alone, fails to establish a utility for the invention of claim 1.

5. However, appellants assert that Sleckman<sup>3</sup> functionally characterized Pb99 as a G-protein coupled receptor. While Sleckman published the same

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<sup>3</sup> Sleckman et al. (Sleckman), “Cloning and Functional Characterization of the Early-Lymphocyte-Specific Pb99 Gene,” *Molecular and Cellular Biology*, Vol. 20, No. 12, pp. 4405-4410 (2000). Brief, Exhibit G.

year as appellants' filing date, we disagree with appellants that Sleckman characterized Pb99 as a G-protein coupled receptor. Therefore, we disagree with appellants' assertion that Sleckman supports a conclusion that the sequence of claim 1 must encode a G protein-coupled receptor because it shares some degree of similarity with Pb99. Request, page 2.

Sleckman reports on the cloning and functional characterization of an early-lymphocyte-specific gene - Pb99. See e.g., Sleckman title. In this report, Sleckman mentions G protein-coupled receptors three times. First, Sleckman's abstract states “[t]he cDNA with the longest open reading frame encodes a putative protein that has seven hydrophobic domains similar to those of seven membrane-spanning proteins, such as the classical G protein-coupled receptors.” We do not find this statement to be a characterization of Pb99 as a G protein-coupled receptor. To the contrary, Sleckman is simply noting that Pb99 has seven hydrophobic domains which are similar to those in seven membrane-spanning proteins, for example, G protein-coupled receptors. Similarly, in the second column of page 4406, Sleckman restates that Pb99 “may be a seven-membrane-spanning protein similar to G protein-coupled receptors . . . .” In our opinion, this is far from a functional characterization of Pb99 as a G protein-coupled receptor. Finally, Sleckman states that Pb99 “contains a hydrophobic signal peptide and seven distinct hydrophobic domains, suggesting that it may be an integral membrane protein that spans the membrane seven times, similar to the classical G protein-coupled receptors. . . .” Sleckman, page 4409, second column. Again, it is our opinion that this is far from a functional characterization of Pb99 as a G protein-coupled receptor.

Instead of functionally characterizing Pb99 as a G protein-coupled receptor, Sleckman recognizes that “Pb99 shares some homology with members of the recently described EGF-TM7 subfamily of cell surface receptors . . . .” Id. According to Sleckman, “it has been proposed that the EGF-TM7 proteins are receptors which function in the immune response . . . . However, a clear role for the EGF-TM7 receptors in the immune response has yet to be elucidated.”

Sleckman, page 4409, column 2. Lastly, Sleckman concludes that “[f]uture detailed analysis of the Pb99-deficient mice will further elucidate the role of the Pb99 protein in lymphocyte function and development . . .” Id. Thus, contrary to appellants’ assertion, Sleckman does not functionally characterize Pb99 as a G protein-coupled receptor; instead, Sleckman expressly states that further research is necessary to elucidate the role of the Pb99 protein.

In addition, regarding Sleckman’s recognition (page 4409, second column) that Pb99 has seven distinct hydrophobic domains, suggesting that it may span the membrane seven times, we note that Ji teaches (page 17299, column 1), “there are putative seven transmembrane molecules, which do not appear to be coupled to a G protein.” Accordingly, we are not persuaded by appellants’ assertion that Sleckman functionally characterizes Pb99 as a G protein-coupled receptor.

For the foregoing reasons, we disagree with appellants’ assertion “that the present case directly tracks Example 10 of the Revised Interim Utility Guidelines Training Materials . . .” Request, bridging sentence, pages 2-3, emphasis removed. Unlike the facts in this case, the facts set forth in Example 10 of the Training Materials establish that the sequence disclosed therein encodes a DNA ligase which has a well-established use in the art. On this record, there is no evidence that the claimed sequence is a G protein-coupled receptor. Further, for the reasons set forth in the Decision, even if the claimed nucleic acid encodes a G protein-coupled receptor, there is no disclosed utility for this receptor. In this regard, we note that claim 1 encompasses a 22 base pair fragment of SEQ ID No:43. There is no evidence on this record that this 22 base fragment of SEQ ID No:43 will encode a function protein.

On reflection, for the reasons set forth above, in addition to those set forth in the Decision, we reaffirm the rejection of claim 1 under 35 U.S.C. § 101 as lacking utility and § 112, first paragraph, for lack of enablement based on the finding of lack of utility. Claims 2, 3 and 6-9 fall together with claim 1.

We have carefully reviewed the original opinion in light of appellants' request, but we find no point of law or fact which we overlooked or misapprehended in arriving at our decision. Therefore, appellants' request has been granted to the extent that the decision has been reconsidered, but such request is denied with respect to making any modifications to the decision affirming the rejection under 35 U.S.C. § 101 as lacking utility and § 112, first paragraph, for lack of enablement based on the finding of lack of utility.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a).

REHEARING DENIED

  
Toni R. Scheiner )  
Administrative Patent Judge )  
  
  
Donald E. Adams ) BOARD OF PATENT  
Administrative Patent Judge )  
  
  
Eric Grimes )  
Administrative Patent Judge ) APPEALS AND  
INTERFERENCES

DEA/lbg

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